Nodal signaling regulates endodermal cell motility and actin dynamics via Rac1 and Prex1.

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Public Summary:

Scientific Abstract:
Embryo morphogenesis is driven by dynamic cell behaviors, including migration, that are coordinated with fate specification and differentiation, but how such coordination is achieved remains poorly understood. During zebrafish gastrulation, endodermal cells sequentially exhibit first random, nonpersistent migration followed by oriented, persistent migration and finally collective migration. Using a novel transgenic line that labels the endodermal actin cytoskeleton, we found that these stage-dependent changes in migratory behavior correlated with changes in actin dynamics. The dynamic actin and random motility exhibited during early gastrulation were dependent on both Nodal and Rac1 signaling. We further identified the Rac-specific guanine nucleotide exchange factor Prex1 as a Nodal target and showed that it mediated Nodal-dependent random motility. Reducing Rac1 activity in endodermal cells caused them to bypass the random migration phase and aberrantly contribute to mesodermal tissues. Together, our results reveal a novel role for Nodal signaling in regulating actin dynamics and migration behavior, which are crucial for endodermal morphogenesis and cell fate decisions.